HEART FAILURE*

(ABSTRACT)**

ARTHUR M. FISHBERG
Associate Physician, Beth Israel Hospital
New York

The presentation is devoted to data available for clinical differentiation of individual types of circulatory failure. The basis of classification is the nature of the disturbance in the *dynamics* of the circulation. As a preliminary, some of the means by which the heart accommodates itself to increased work are discussed.

DILATATION OF THE HEART

In at least a very high proportion of instances, dilatation of the heart is a useful process, playing a fundamental part in the adaptation of the heart to increased work. The studies of Starling, Frank, Straub and others have shown that when either heightened arterial resistance or greater venous inflow increases the work of the heart, accommodation to the greater load involves increase in diastolic volume. When the initial (diastolic) length of the muscle fiber is increased, more energy is liberated in the succeeding systole. The compensatory significance of dilatation of the appropriate chambers of the heart in arterial hypertension, valvular defects, etc., thus becomes clear. So-called myogenous dilatation (in myocarditis, etc.) is discussed, and it is concluded that in this form there is also a compensatory element.

HYPERTROPHY OF THE HEART

Hypertrophy follows dilatation. The link that connects hypertrophy with antecedent dilatation is not clear, but

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the utility of the process is elucidated by the following considerations: Inasmuch as the volume of a sphere is proportional to the cube of the radius, as a sphere increases in size, equal increments in volume correspond to smaller and smaller increases in radius. In other words, the larger a cardiac chamber, the less its radius must be diminished to discharge the same volume of blood. Thus, the fibers of the dilated heart contract a shorter distance in maintaining the same stroke volume than do those of the normal organ. But the force of contraction must be correspondingly greater. Dilatation thus leads to the necessity for a shorter but more powerful contraction of the muscle fibers, and hypertrophy would seem to be the adaptation to these altered conditions. Of course, such a line of thought merely considers the wherefore and not the how of the process by which hypertrophy occurs in the dilated heart.

Increase in Rate

The conditions for tachycardia unfolding a compensatory action are especially favorable in cardiac failure, for the shortening of diastole that accompanies tachycardia is neutralized to a large extent by the increased venous pressure, which accelerates diastolic filling. In auricular fibrillation the loss of the quota due to auricular systole militates against compensation by tachycardia.

Among the factors that may play a part in the pathogenesis of tachycardia in cardiac failure is the Bainbridge reflex, the acceleration in rate due to increased pressure near the mouths of the venae cavae and the auricle. With sharp fall in blood pressure, reflexes originated in the carotid sinus and aorta may also be concerned.

The Cardinal Circulatory Syndromes

The primary distinction is between circulatory failure of cardiac and of peripheral origin. In the category of cardiac failure, further classification on a dynamic basis is often feasible for the clinician. In certain cases, the

cardiac failure is obviously a result of interference with diastole, either in consequence of mechanical incarceration of the heart or because of undue shortening of diastole in tachycardia. These cases may be termed *diastolic failure.* Far more common is cardiac insufficiency where there is no hindrance to diastolic filling but contractility is inadequate. Such *systolic failure* may be confined to the right or the left ventricle while the other chamber is functionally efficient—right or left ventricular failure. The peripheral insufficiencies are doubtless of variegated nature, but they cannot as yet be clinically differentiated.

The syndromes are considered in the following order:

1. Cardiac insufficiencies.
   - A. Systolic insufficiencies—impaired contractility
     - a. Failure of the left ventricle
     - b. Failure of the right ventricle
   - B. Diastolic insufficiencies—inadequate diastolic filling.
     - a. Mechanical incarceration of the heart
     - b. Abbreviation of diastole by tachycardia
2. Peripheral insufficiencies—deficient venous return to the heart
3. Combinations of the above.

Failure of the Left Ventricle

Failure of the left ventricle is seen in arterial hypertension, disease of the aortic valve, sclerosis of the left coronary artery, and mitral disease with predominant regurgitation. In acute glomerulo-nephritis, left ventricular failure is the chief danger in the first days of the disease.

The clinical picture of isolated insufficiency of the left ventricle is characterized by symptoms and signs attributable to increased tension in the pulmonary circuit in the presence of normal pressure in the systemic veins. The dominant symptom is dyspnea; often, there are attacks of

nocturnal cardiac asthma. Despite the presence of arterial hypertension, the pulmonic second sound is apt to be the louder. The liver is not enlarged and there is no edema. The stage of isolated insufficiency of the left ventricle may last for many years. Such patients are not uncommonly thought to suffer from bronchial asthma. While some succumb during the stage of isolated failure of the left ventricle, most ultimately develop right ventricular failure.

The dynamic disturbances and symptomatology of mitral stenosis may closely resemble that of left ventricular failure except that nocturnal cardiac asthma is unusual. Here, the circulatory disturbance is really left auricular failure.

Failure of the Right Ventricle

Right ventricular failure occurs in conditions with increased tension in the pulmonary circuit, i. e., mitral disease, emphysema, various forms of pulmonary fibrosis, kyphoscoliosis, extensive pleural adhesions, the rare forms of disease of the pulmonary artery described by Ayerza, etc. Unusual causes are organic changes in the pulmonary and tricuspid valves and disease of the right coronary artery. The superimposition of right ventricular failure on that of the left ventricle was mentioned above.

Failure of the right ventricle is documented by increase in pressure in the systemic veins and swelling of the liver. Cardiac edema is another prime symptom. Cyanosis and dyspnea are almost always present, but in contradistinction to left ventricular failure, the cyanosis is relatively more intense than the dyspnea. The causes of this phenomenon are discussed. Somnolence may be a prominent symptom in severe cases. The great clinical importance of measurement of venous pressure is emphasized. Libman's syndrome of acute occlusion of the right coronary artery—rapid, intense engorgement of the liver combined with sino-auricular block—is mentioned.

Cardiac Failure Due to Inadequate Diastolic Filling

Two types of cardiac failure due to inadequate diastolic filling are discussed: 1. Mechanical limitation of diastole due to pericardial effusion or adhesive mediastino-pericarditis with shrinking; and 2. Shortening of diastole in excessive tachycardia.

Circulatory Failure of Peripheral Origin

The peripheral failures are due to stagnation of blood in the periphery of the circulation so that the venous return to the heart is diminished. An important feature for the differentiation from failure of the right heart is the low venous pressure in the peripheral failures. Peripheral circulatory failures occur in surgical and traumatic shock, many of the acute fevers, and diabetic acidosis, to mention only the most common.

The circulatory disturbance resulting from diminished venous return to the heart is one for which the heart cannot compensate. The reason for this is that filling is an entirely passive process on the part of the heart, which functions solely as a force pump; it does not, like a suction pump, aspirate blood from the venae cavae.

There are probably various pathogenetically different types of peripheral failure. Among the factors that have been considered as significant in the causation of peripheral failure are diminution in tonus of the small vessels with resultant increased capacity, defective function of the mechanisms participating in the return of blood to the heart (Henderson's venopressor mechanism), and diminution in circulating blood volume.

The importance of peripheral failure in the clinical picture of coronary thrombosis is discussed. In such cases, the venous pressure is often low, testifying to the peripheral pooling of the blood, which serves to protect the acutely damaged heart.